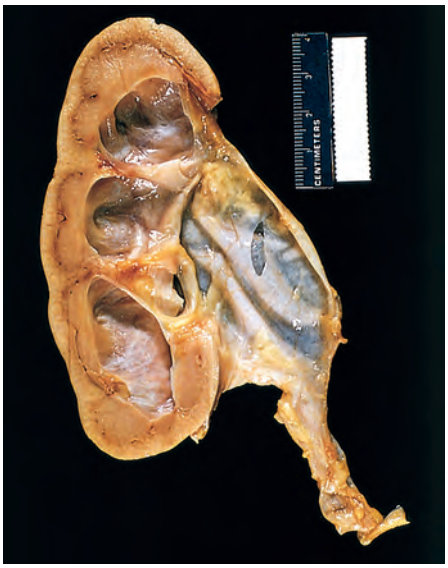

Figure 20-48 Hydronephrosis of the kidney, with marked dilation of the pelvis and calyces and thinning of the renal parenchyma.

capsule. Most of the early symptoms are produced by the underlying cause of the hydronephrosis. Thus, calculi lodged in the ureters may give rise to renal colic, and prostatic enlargements may give rise to bladder symptoms.

Unilateral complete or partial hydronephrosis may remain silent for long periods, since the unaffected kidney can maintain adequate renal function. Sometimes its existence first becomes apparent in the course of imaging studies. In its early stages, perhaps the first few weeks, relief of obstruction leads to reversion to normal function. *Ultrasonography* is a useful noninvasive technique in the diagnosis of obstructive uropathy.

In *bilateral partial obstruction* the earliest manifestation is inability to concentrate urine, reflected by polyuria and nocturia. Some patients develop distal tubular acidosis, renal salt wasting, secondary renal calculi, and chronic tubulointerstitial nephritis with scarring and atrophy of the papilla and medulla. Hypertension is common.

Complete bilateral obstruction of rapid onset results in oliguria or anuria and is incompatible with survival unless the obstruction is relieved. Curiously, after relief of complete urinary tract obstruction, postobstructive *diuresis* occurs. This can often be massive, with the kidney excreting large amounts of urine that is rich in sodium chloride.

Urolithiasis (Renal Calculi, Stones)

Urolithiasis affects 5% to 10% of Americans in their lifetime and the stones may form anywhere in the urinary tract, but most arise in the kidney. Men are affected more often than women, and the peak age at onset is between 20 and 30 years. Familial and hereditary predisposition to stone formation has long been known. Many inborn errors of metabolism, such as cystinuria and primary hyperoxaluria, provide examples of hereditary disease characterized by excessive production and excretion of stone-forming substances.

Etiology and Pathogenesis. There are four main types of calculi (Table 20-12): (1) *calcium stones* (about 70%), composed largely of calcium oxalate or calcium oxalate mixed with calcium phosphate; (2) another 15% are so-called *triple stones* or *struvite stones*, composed of magnesium ammonium phosphate; (3) 5% to 10% are *uric acid stones*; and (4) 1% to 2% are made up of cystine. An organic mucoprotein matrix, making up 1% to 5% of the stone by weight, is present in all calculi. **Although there are many causes for the initiation and propagation of stones, the most important determinant is an increased urinary concentration of the stones' constituents, such that it exceeds their solubility (supersaturation).** A low urine volume in some metabolically normal patients may also favor supersaturation.

- *Calcium oxalate stones* (Table 20-12) are associated in about 5% of patients with hypercalcemia and hypercalciuria, such as occurs with hyperparathyroidism, diffuse bone disease, sarcoidosis, and other hypercalcemic states. About 55% have hypercalciuria without hypercalcemia. This is caused by several factors, including hyperabsorption of calcium from the intestine (absorptive hypercalciuria), an intrinsic impairment in renal tubular reabsorption of calcium (renal hypercalciuria), or idiopathic fasting hypercalciuria with normal parathyroid function. As many as 20% of calcium oxalate stones are associated with increased uric acid secretion *(hyperuricosuric calcium nephrolithiasis)*, with or without hypercalciuria. The mechanism of stone formation in this setting involves "nucleation" of calcium oxalate by uric acid crystals in the collecting ducts. Five percent are associated with *hyperoxaluria*, either hereditary (primary oxaluria) or, more commonly, acquired by intestinal overabsorption in patients with enteric diseases. *Hypocitraturia,* which can be idiopathic or associated with acidosis and chronic diarrhea of unknown cause, may produce calcium stones. In a variable proportion of

Table 20-12 Prevalence of Various Types of Renal Stones

Stone Type	Percentage of All Stones
Calcium Oxalate and Phosphate	70
Idiopathic hypercalciuria (50%)	
Hypercalciuria and hypercalcemia (10%)	
Hyperoxaluria (5%)	
Enteric (4.5%)	
Primary (0.5%)	
Hyperuricosuria (20%)	
Hypocitraturia	
No known metabolic abnormality (15% to 20%)	
Magnesium Ammonium Phosphate (Struvite)	5-10
Uric Acid	5-10
Associated with hyperuricemia	
Associated with hyperuricosuria	
Idiopathic (50% of uric stones)	
Cystine	1-2
Others or Unknown	±5